Inferences about Respiratory Muscle Use after Cardiac Surgery from Compartmental Volume and Pressure Measurements

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Background: After upper abdominal surgery, patients have been observed to have alterations in respiratory movements of the diaphragm and abdominal and respiratory shifts in pleural and abdominal pressure that suggest dysfunction of the diaphragm. The validity of such deductions about diaphragm function from these observations is open to discussion.

Methods: In eight adult patients, American Society of Anesthesiologists physical status 2, scheduled for elective cardiac surgery, we measured respiratory rate, tidal volume, rib cage and abdominal cross-section changes, and esophageal (Poe) and gastric (Pga) pressures preoperatively, 1 day postoperatively, and 5 days postoperatively. These data were analyzed in detail by following the variables through each respiratory cycle.

Results: Mean ΔPoe/ΔPga decreased from 0.75 preoperatively to −0.56 1 day postoperatively and recovered to 0.47 5 days postoperatively. Plots of Poe against Pga and rib cage against abdominal expansion (Konno-Mead diagrams) were constructed. Six patients showed a postoperative pattern of breathing similar to that seen in patients who have undergone abdominal surgery: a decrease in the ratio of ΔPoe/ΔPga, and a shift toward rib cage expansion, with an increase in breathing rate and a decrease in tidal volume. This change was accomplished in most cases by the use of abdominal muscles in expiration with an increase in inspiratory intercostal muscle action without an increase in diaphragm activation, that is, a shift in the normal balance of respiratory muscle use in favor of muscles other than the diaphragm. A different ventilatory pattern was observed in the other two patients, consisting of minimal rib cage excursion and a large abdominal excursion. In these cases tidal volume was generated largely by contraction and relaxation of abdominal muscles with probable redistribution in diaphragm activity. In addition, five patients exhibited positive changes in Pga at the end of inspiration that corresponded to closure of the upper airway, relaxation of inspiratory muscles, and subsequent opening of the airway with sudden exhalation, producing a grunt.

Conclusions: Indirect measurements of respiratory muscle action based on pressure and chest wall motion are easier than are assessments based on implanted electromyogram electrodes and sonomicrometers that measure electric activity and muscle length, respectively, directly. Interpretation requires numerous assumptions and detailed analysis of phase relations among the variables. In patients after thoracic surgery, however, these measurements strongly point to a shift in the distribution of motor output toward muscles other than the diaphragm. (Key words: Lung(s), ventilation: effect of cardiac surgery. Muscle, diaphragm: function after cardiac surgery. Surgery, cardiac: ventilatory changes.)

ABNORMAL behavior of the respiratory muscles has been observed in patients immediately after surgery on the upper abdomen and may contribute to the pathogenesis of postoperative pulmonary complications. These patients breathe with a high respiratory rate and a small tidal volume, maintaining normal minute ventilation. Respiratory movements of the abdomen and changes in gastric pressure (Pga) are reduced. Phasic activity of abdominal expiratory muscles is prominent, and a decrease in diaphragm activity has been suggested. Parallel experiments in dogs after abdominal operations show a marked reduction in the degree of

Materials and Methods

Subjects

Eight male patients with physical status 2 scheduled for cardiac surgery were included (age ± SD) was 4 ± 0.8 years, and weight ± SD) was 77 ± 9 kg. All patients received a constant thoracic epidural anesthetic throughout the period, and no respiration was allowed. Three subjects each had a thoracic epidural catheter placed. The other subjects received general anesthesia. No premedication was given.

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RESPIRATORY MUSCLE USE AFTER CARDIAC SURGERY

...shortening in inspiration.\textsuperscript{5} In other experiments, touching, pulling, or stretching of the peritoneum, gall bladder, or intestine can trigger reflexes that change respiration in much the same way as changes seen after abdominal surgery.\textsuperscript{6,7} The altered pattern of respiration may promote development of abdominal edema and impaired gas exchange.

It is important to know whether respiratory muscle behavior also is abnormal after operations on the thorax, but data available to date have not made the extent to which it is. An experimental study on sheep using sonomicrometers\textsuperscript{8} showed a marked reduction in diaphragm shortening after thoracotomy, similar to the finding in dogs after abdominal surgery.\textsuperscript{5} One study in humans undergoing thoracotomy for pneumonectomy or lobectomy found some changes in rib cage abdomen movement and P\textsubscript{es} and pleural pressure changes but concluded that there was little evidence for any diaphragm dysfunction.\textsuperscript{9} A study in the 2nd week after cardiac surgery found no evidence of diaphragm dysfunction assessed by electric stimulation of the phrenic nerves.\textsuperscript{10} More recently, sonomicrometers placed in human costal hemidiaphragms on the same side as a lobectomy showed reduced shortening and even paradoxical lengthening of that part of the muscle during inspiration.\textsuperscript{11} Sonomicrometers experiments give the most direct and convincing data about local diaphragm function but are difficult to perform in humans and have the potential disadvantage that installation of the devices into the diaphragm may induce a local or general change in diaphragm function.

In the current study we therefore attempted to analyze the use of respiratory muscles in the postoperative period in eight patients undergoing midline sternotomy for operation on the heart, by means of detailed analysis of rib cage and abdominal expansion and of esophageal pressure (P\textsubscript{es}) and P\textsubscript{es}, including their phase relations.

### Materials and Methods

**Subjects**

Eight male American Society of Anesthesiologists physical status 2 patients scheduled for elective cardiac surgery were included in this study. Their age (mean ± SD) was 43 ± 11 yr (range 27–57 yr). Their weight was 77 ± 9 kg (range 68–97 kg). None of them had constant thoracic pain in the immediate preoperative period, and none was taking opioids before the operation. Three patients smoked, but none had clinical evidence of chronic obstructive pulmonary disease, and none was obese. The characteristics of the patients are shown in table 1.

Institutional ethical committee approval was obtained, and all subjects volunteered to take part in the study after the nature and purpose of the experiment were explained to them.

**Rib Cage and Abdominal Dimensions**

Tidal changes in rib cage and in abdominal dimensions were measured simultaneously using a respiratory inductance plethysmograph (RIP) (Respitrace, White Plains, NY). A calibration of RIP measurements to spirometry was performed to calculate volume-motion coefficients for rib cage (VCM\textsubscript{Rc}) and abdomen (VCM\textsubscript{Ab}). Patients were asked to alternate between predominantly abdominal and thoracic breathing, while changes in the cross section of the two compartments were matched to simultaneously measured spirometric volumes (V\textsubscript{T}). From these measurements, a regression line was found by the least squares method between abdominal cross section and V\textsubscript{T} on the x axis and between rib cage cross section and V\textsubscript{T} on the y axis. The intercepts of the regression line with the x axis and y produced 1/VCM\textsubscript{Rc} and 1/VCM\textsubscript{Ab}, respectively. Details concerning the calibration procedure and the calculation of VMC have been reported.\textsuperscript{12} Volume was calculated from the instantaneous sum of the RIP values for abdominal and rib cage volumes. Because cardiac surgery may change the geometric characteristics of the chest wall and result in changes in the VMCs of the

<table>
<thead>
<tr>
<th>Table 1. Characteristics of the Patients and Type of Surgery</th>
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<tr>
<td>Patient No.</td>
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<td>2</td>
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<td>3</td>
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<td>4</td>
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<td>5</td>
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<td>6</td>
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<tr>
<td>7</td>
</tr>
<tr>
<td>8</td>
</tr>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>SD</td>
</tr>
</tbody>
</table>

CPB time = cardiopulmonary bypass time; AVR = aortic valve replacement; CABG = coronary artery bypass grafting surgery; ASD = atrial septal defect closure; VSD = ventricular septal defect closure.
two compartments. Calibration of the RIP was repeated immediately before the measurements made on each day of observation. Spirometric measurements were made with a nose clip and a mouthpiece, connected to a heated number 2 Fleisch pneumotachograph, connected to a differential pressure transducer and integrator (17212, Godart, Bilthoven, The Netherlands).

The results of each set of measurements are the means of all cycles during a 5-min period of quiet breathing. Cross-section changes and VMC calculation of the abdominal and rib cage tidal volumes (V_A and V_R, respectively) were used to calculate abdominal and rib cage contributions to tidal breathing (V_A/V_T and V_R/V_T). Minute ventilation was obtained by the product of the RIP sum tidal volume (V_T) and respiratory rate.

**Gastric and Esophageal Pressures**

P_{ga} and P_{es} were used as indexes of pleural and abdominal pressure changes. They were measured using two thin-walled 10-cm-long balloons connected to polyethylene tubing (120 cm long and 1.7 mm ID), which were positioned in the middle third of the esophagus and in the stomach and filled with 0.5 and 1 ml air, respectively. Each balloon was connected to a pressure transducer (DP15, Valdely, Northridge, CA). Transdiaphragmatic pressure (P_{di}) was calculated later by subtraction (P_{di} = P_{ga} - P_{es}) using instantaneous values of P_{ga} and P_{es}.

Changes in P_{ga} and P_{es} were measured during inspiration by subtracting the pressure measured during the last 100 ms of the inspiratory phase from the peak positive or negative pressure developed during inspiration, determined on the rib cage tracing of the RIP, as previously reported by Duggan and Drummond. All values presented for P_{ga} and P_{es} represent the means of all respiratory cycles recorded during a 5-min period of quiet breathing. The postoperative period, the stomach was emptied before each set of measurements using a gastric suction tube inserted and removed before the ventilatory measurements. Many patients had "grunting" respiration (described in Results and Discussion) on the 1st postoperative day. Sections of the record where this occurred were avoided in the quantitative analysis.

Changes in cross-section dimensions, in P_{ga}, P_{es}, and P_{es} were recorded on a strip chart recorder (ES 1000, Gould, Valley View, OH). All values were corrected to body temperature, pressure, saturated.

**Experimental Procedure**

All of the patients were studied while in the supine 30° head-up position. Preoperative baseline ventilatory measurements were performed the day before surgery. Anesthetic agents used during surgery included diazepam 25-50 mg, fentanyl 1.6-4 mg, and pancuronium 14-25 mg. All patients had a median sternotomy. The surgical procedure consisted of a coronary artery bypass graft in five patients, an atrial septal defect closure in one patient, a ventricular septal defect closure in one patient, and an aortic valve replacement in one patient. During cardiopulmonary bypass, myocardial preservation was achieved in all patients by systemic cooling, cold cardioplegia, and topical cooling of the heart with iced saline slush.

At each set of measurements, after the calibration procedure of the RIP, patients were left in a quiet environment without nose clip or mouthpiece for 10-15 min to obtain a steady-state breathing pattern. They were fully conscious throughout the measurements and were able to breathe quietly comfortably without pain, although many felt pain on maximal breathing maneuvers. No opioid analgesics were used in the postoperative period. Pain was controlled by nonsteroidal anti-inflammatory drugs.

**Statistical Analysis**

All values are reported as means ± SD. Comparisons between the periods of measurements were performed by a two-way analysis of variance for repeated measurements and a Newman Keuls test for multiple comparisons, when appropriate. A P value less than 0.05 was considered significant.

**Results**

Group data (table 2) show changes in RIP sum tidal volume, respiratory rate, minute ventilation, rib cage and abdominal expansion, and changes in P_{es} and pleural pressure rather similar to those reported in series of patients after upper abdominal surgery. There was a shift to rapid shallow breathing on the 1st postoperative day with no significant change in minute ventilation. The V_A/V_T decreased, without a change in V_R. Inspiratory P_{es} changes were smaller, actually becoming negative in five of the eight patients, and the ratio of the maximum change in P_{es} from the end-expiratory point during inspiration and the maximum change in P_{ga} (ΔP_{ga}/ΔP_{es}) decreased. When the pattern of breathing was examined in terms of plots of P_{ga} against time, it was noted that the patients were unable to increase P_{ga} with the abdominal expiratory effort during inspiration (fig. 5).

The other two patients were unable to increase P_{ga} with the abdominal expiratory effort during inspiration. The changes in P_{ga} and P_{es} are shown in detail below.

Average data for all patients during the respiratory relaxation period showed similar to those of the patients in this study. Changes in P_{ga} and P_{es} during expiration and inspiration were calculated. Rate of increase in P_{ga} during expiration and rate of decrease in P_{es} during inspiration for these six postoperative patients were calculated.

### Table 2. Result of RIP Sum Tidal Volume During Inspiration

<table>
<thead>
<tr>
<th>V_A</th>
<th>T</th>
<th>V_A/V_T</th>
<th>ΔP_{ga}</th>
<th>ΔP_{es}</th>
<th>ΔP_{di}</th>
<th>T</th>
<th>ΔP_{di}/ΔP_{es}</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.23</td>
<td>1.15</td>
<td>0.90</td>
<td>1.27</td>
<td>0.89</td>
<td>0.39</td>
<td>0.05</td>
<td>0.43</td>
</tr>
<tr>
<td>1.23</td>
<td>1.15</td>
<td>0.90</td>
<td>1.27</td>
<td>0.89</td>
<td>0.39</td>
<td>0.05</td>
<td>0.43</td>
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</tbody>
</table>

* P < 0.01

Table 2. Results of the Different Ventilatory Variables before Surgery and on Days 1 and 5 after Surgery in the Eight Patients, in the Six Patients Detailed in Figure 1, and in the Two Patients Who Experienced a Decrease in Transdiaphragmatic Pressure during Inspiration

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperation</th>
<th>Day 1</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>$Vi$</td>
<td>7.5 ± 2.6.</td>
<td>7.4 ± 2.0</td>
<td>9.2 ± 3.6</td>
</tr>
<tr>
<td>$f$</td>
<td>16.3 ± 4.6</td>
<td>23.6 ± 5.1*</td>
<td>22.2 ± 3.9*</td>
</tr>
<tr>
<td>$V_{SUM}$</td>
<td>482 ± 140</td>
<td>319 ± 61</td>
<td>413 ± 132</td>
</tr>
<tr>
<td>$V_{RC}$</td>
<td>252 ± 93</td>
<td>209 ± 88</td>
<td>261 ± 97</td>
</tr>
<tr>
<td>$V_{BB}$</td>
<td>230 ± 114</td>
<td>110 ± 90†</td>
<td>152 ± 82</td>
</tr>
<tr>
<td>$V_{RC}/V_{SUM}$</td>
<td>0.53 ± 0.14</td>
<td>0.68 ± 0.23</td>
<td>0.83 ± 0.12</td>
</tr>
<tr>
<td>$Ap_g$</td>
<td>2.6 ± 0.9</td>
<td>2.0 ± 2.7†</td>
<td>2.2 ± 1.0</td>
</tr>
<tr>
<td>$Ap_{Pes}$</td>
<td>3.5 ± 0.8</td>
<td>4.8 ± 2.6</td>
<td>4.7 ± 1.1</td>
</tr>
<tr>
<td>$Ap_{di}$</td>
<td>6.1 ± 1.6</td>
<td>2.3 ± 4.7</td>
<td>6.2 ± 2.8</td>
</tr>
<tr>
<td>$Ap_{g}/Ap_{Pes}$</td>
<td>0.73 ± 0.22</td>
<td>0.51 ± 1.1†</td>
<td>0.47 ± 0.20</td>
</tr>
<tr>
<td>$T_{i}$</td>
<td>1.45 ± 0.43</td>
<td>1.04 ± 0.19</td>
<td>—</td>
</tr>
<tr>
<td>$Ap_{Pes}/T_{i}$</td>
<td>2.5 ± 0.7</td>
<td>5.1 ± 3.5</td>
<td>2.5 ± 0.8</td>
</tr>
<tr>
<td>$Ap_{di}/T_{i}$</td>
<td>4.3 ± 1.2</td>
<td>4.2 ± 2.4</td>
<td>2.4 ± 1.4</td>
</tr>
<tr>
<td>$V_{i}/V_{f}$</td>
<td>0.15 ± 0.1</td>
<td>0.09 ± 0.05</td>
<td>—</td>
</tr>
</tbody>
</table>

* $P < 0.01$; † $P < 0.05$

Average data for the first group show changes in $Vi$, respiratory rate, minute ventilation, $V_{RC}$, and $P_{aw}/P_{aw}$ similar to those for the whole set of eight patients. For these six, the change in $P_{aw}$ averaged 5.6 cm H2O preoperatively and 4.8 cm H2O on the 1st day after surgery. Rate of increase in $P_{aw}$ (change in $P_{aw}$ between end-expiration and end-inspiration per change in inspiratory time taken from rib cage excursion) was 4.2 cm H2O/s preoperatively and 5.0 cm H2O/s postoperatively. The ratio of $\Delta P_{aw}$ to RIP sum tidal volume, a rough estimate of pulmonary impedance, was 6.3 cm H2O/ml preoperatively and 12.3 cm H2O/ml postoperatively. $V_{RC}/V_{f}$ changed from 0.54 preoperatively to 0.78 postoperatively.

The other two patients showed a very unusual pattern of breathing on the 1st postoperative day, corresponding to the rib cage-abdomen and $\Delta P_{aw}/\Delta P_{aw}$ plots in figure 3 and the polygraph tracings in figure 4. $P_{aw}$ decreased from the beginning of inspiration, reaching its lowest level just beyond the beginning of expiration, and then increased through expiration and leveled off at end-expiration. In subject 8, there was a small superimposed positive deflection in $P_{aw}$ at end-expiration. Both of these patients expanded their rib cage less than preoperatively. Subject 8 reduced his rib expansion to nearly zero. Subject 7 did not move his rib cage for the first half of inspiration but let it expand in the second half.

Several patients (subjects 3, 6, 7, and 8) in many breaths on the 1st postoperative day showed a peculiar pattern of $P_{aw}$ in the loops shown in figure 1 and 3 and the example shown in figure 5. After an initial negative deflection through inspiration, $P_{aw}$ swung positive in early expiration, to as great as 50 cm H2O in some patients. The positive change in $P_{aw}$ was often accompanied by a movement on the rib cage-abdomen plot in an isovolume direction: rib cage out and abdomen in. The positive waves in $P_{aw}$ were variable from breath to breath but occurred in most breaths postoperatively in three patients, not at all in three patients, and intermittently in the others.

The patterns of breathing in all eight patients had reverted toward normal by the 5th postoperative day, although respiratory frequency remained high, and
of atelectasis or elevated diaphragm typical of severe cold block diaphragm dysfunction. Transient diaphragm paresis related to the cold block could not be excluded, however.

Discussion

Postoperative Diaphragm Dysfunction

Results very similar to those shown in table 2 have been reported in several studies of patients after upper abdominal surgery and by Maeda et al. in a group of 21 patients after thoracotomy for pneumonectomy or lobectomy.\(^6\) In the earlier studies,\(^3\) the data were interpreted to indicate a substantial decrease in diaphragm activity on the 1st postoperative day but that interpretation has been challenged. Estimates of diaphragm contribution to inspiration were based on the relation between changes in P\(_{al}\) and pleural pressure according to the theory of Macklem et al.\(^7\) for inspirations from relaxed functional residual capacity. More recently a study of chest wall mechanics has modified these concepts,\(^8\) and the recognition that abdominal muscle contraction may occur in patients after surgery has required a reinterpretation of pressure-volume curve.

A decrease in breathing rate is considered to indicate that other factors, such as surgery can affect diaphragm contractility. Problems in the interpretation of the changes in P\(_{al}\) and a change in P\(_{di}\) for inspiration from the abdomen patients postoperatively are not abnormal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation. In our patients, the minimum change in P\(_{al}\) was associated with the operation of the surgical wound or the change in P\(_{di}\) from inspiratory activity and normal ventilation.

Likewise, the presence of upper abdominal insufflation, which is normal decreases P\(_{al}\) to breathing. This effect, in part to increase

![Fig. 1. Rib cage volume (RC) and abdominal volume (ABD) tracings from respiratory inductance plethysmograph signals (top) and tracings of changes in esophageal and gastric pressures (ΔPes and ΔPga, respectively) (bottom) from each of six patients during quiet breathing on the preoperative, 1st, and 5th postoperative days. Pressure and volume calibrations represent 1 cmH\(_2\)O and 100 ml, respectively. The preoperative loops cannot be superimposed accurately on the postoperative loops because the values of volumes and pressures at end-expiration are not known. Because of abdominal muscle activity, the postoperative end-expiratory points on the rib cage-abdomen plot must be some distance to the left of the preoperative position, and on the ΔPga/ΔPes plot, must be to the right, but the distance and direction cannot be determined accurately. Solid line = inspiration; dotted line = expiration.](image)

![Fig. 2. Interpretation of the relations between esophageal pressure (P\(_{es}\)) and gastric pressure (P\(_{ga}\)) (plots in fig. 1). Negative P\(_{es}\) is plotted on the ordinate and positive P\(_{es}\) on the abscissa. Lines of iso-transdiaphragmatic pressure (iso-P\(_{al}\)) (iso-P\(_{es}\) = P\(_{es}\)) are downward-sloping 45° diagonals (dashed lines). Movement on the plot upward and to the right crosses iso-P\(_{es}\) lines in the direction of increasing transdiaphragmatic pressure (P\(_{al}\)). Movement along a dashed line implies no change in P\(_{al}\).](image)

![Fig. 3. Rib cage volume (RC) and abdominal volume (ABD) tracings from respiratory inductance plethysmograph signals (top) and changes in esophageal and gastric pressures (ΔPes and ΔPga, respectively) (bottom) from each of six patients during inspiration and expiration. Subject 7: right and left lung ventilation; dotted line 1 = inspiration; dotted line 2 = expiration.](image)
RESPIRATORY MUSCLE USE AFTER CARDIAC SURGERY

Figure 3. Rib cage volume (RC) and abdominal volume (ABD) tracings from respiratory inductance plethysmograph signals (top) and tracings of changes in esophageal and gastric pressures (ΔPes and ΔPga, respectively) (bottom) on the 1st postoperative day from each of the two patients in whom a decrease in transdiaphragmatic pressure developed during inspiration (negative ΔPga). (Left) Subject 7; (right) subject 8. Solid line = inspiration; dotted line = expiration; distance between dots = 250 ms.

Figure 4. Esophageal and gastric pressures and transdiaphragmatic pressure (Pdi) tracings (positive up), and tracings of rib cage and abdominal volumes (C and ABD, respectively) from respiratory inductance plethysmograph signals from subjects 7 (left) and 8 (right) on the 1st postoperative day. The Pdi tracing shows a negative change in Pdi during inspiration while ABD is positive, suggesting the release of the passive stretch of the diaphragm when abdominal muscles relax. In subject 7, the decrease is followed by an increase in Pdi beginning in midexpiration while ABD continues to increase, suggesting an end-inspiratory active contraction of the diaphragm superimposed on relief of passive stretch.

A decrease in the ratio \( \Delta P_{\text{es}} / \Delta P_{\text{es}} \) was originally considered to indicate a decrease in diaphragmatic activity, but other factors in patients who have undergone surgery can affect this ratio. First, by relaxing as the diaphragm contracts, abdominal muscles tend to decrease the changes in Pdi, and can even shift the maximum change in Pdi from the end-expiratory point during inspiration from positive to negative. Second, after surgery, patients probably have an increase in the overall drive to breathe, suggested by an increase in occlusion pressure, an increase in the work of breathing, and normal ventilation despite an increase in lung impedance. In our patients, for example the ratio of the maximum change in Pdi to Vt, which gives a rough indication of impedance, increased from 6.3 preoperatively to 13.3 ml/cmH₂O postoperatively. When overall drive to breathe increases, intercostal and accessory muscle activity increases proportionately more than diaphragm activity, which tends to decrease the ratio \( \Delta P_{\text{es}} / \Delta P_{\text{es}} \).

Likewise, the increase in the ratio of rib cage to abdominal expansion does not necessarily imply an abnormal decrease in the contribution of the diaphragm to breathing. This increased ratio may be attributable in part to increased intercostal inspiratory activity as has required a more complicated analysis of the pressure-volume data.

Anesthesiology. V 82, No 6, Jun 1995
sociated with increased overall drive to breathe. In addition, after surgery some patients show tonic activity of abdominal muscles in inspiration,†† which makes the abdominal wall less extensible and enhances the ability of the diaphragm to expand the rib cage. 20

RESPIRATORY MUSCLE USE AFTER CARDIAC SURGERY

Diaphragm Activity
We considered whether an estimate of diaphragm activity in isolation could be made from $P_{io}$. In the six patients, changes in $P_{io}$ averaged 5.6 cmH$_2$O before the operation and 4.8 cmH$_2$O on the 1st day after surgery. Part of this difference is explained by the change in respiratory frequency. The shorter inspiratory time means that even with the same inspiratory flow rate and the same rate of increase of inspiratory electromyographic activity, the value of $P_{io}$ reached by the end of the shortened inspiration must be less than before the operation. An approximate correction can be made for this by using the mean rate of increase in $P_{io}$ in inspiration (change in $P_{io}$ between end-expiration and end-inspiration per change in inspiratory time taken from rib cage excursion), which was 4.2 cmH$_2$O/s before and 5.0 cmH$_2$O/s after the operation (not significantly different). The rate of increase in $P_{io}$ does not change significantly because peak inspiratory $P_{io}$ depends not only on the level of activation of the diaphragm but also on the length of the diaphragm at end-inspiration. Because of abdominal muscle contraction, the end-inspiratory configuration of the chest wall postoperatively has a relatively smaller abdominal volume and a relatively longer diaphragm, implying that $P_{io}$ overestimates the degree of diaphragm activation postoperatively compared with preoperatively. Correction for this force-length effect cannot be estimated accurately. In normal humans, however, it is possible to double the $P_{io}$ obtained for a given level of electric activation of the diaphragm by contracting abdominal muscles and lengthening the diaphragm. Overall, it is hard to be certain that there is any important change in activation of the diaphragm in any of the subjects except 7, and 8, in whom diaphragm activation is probably reduced considerably (see below).

Inspiratory Intercostal and Accessory Muscle Activity in Subjects 1–6
Consideration of the balance of forces acting on the rib cage can be used to estimate activity of the inspiratory intercostal and accessory muscles postoperatively. Volume of the rib cage at end-inspiration depends on the balance between elastic recoil of the rib cage, pleural pressure, the expanding force exerted by the diaphragm and the expanding force exerted by inspiratory intercostal and accessory muscles. In the postoperative period, pleural pressure tended to be more negative (end-inspiratory $P_{io}$ was $-4.8$ cmH$_2$O compared with $-3.5$ cmH$_2$O preoperatively), a condition that should reduce rib cage volume. Inspiratory increase in diaphragm tension, estimated by $P_{io}$, was not statistically changed (the change in $P_{io}$ was $4.8$ cmH$_2$O compared with $5.6$ cmH$_2$O preoperatively), but the effectiveness of the diaphragm at expanding the rib cage will have been greatly impaired because the normal positive inspiratory wave in $P_{io}$ had disappeared (the maximum change in $P_{io}$ from the end-inspiratory point during inspiration was $-2.0$ cmH$_2$O postoperatively compared with $2.0$ cmH$_2$O preoperatively). Positive $P_{io}$ pushes directly outward on the rib cage in the area of apposition and also provides resistance to diaphragm descent so that tension in diaphragm fibers inserted into the rib cage margin tends to elevate the ribs. Despite changes in measured pressure that should reduce its movement, the rib cage expands normally in the patients after surgery. This can best be explained by an increase in inspiratory force exerted by intercostal and accessory muscles (although another possibility is that surgery somehow caused a substantial increase in rib cage compliance).

Abdominal Muscle Activity in Subjects 1–6
Altogether, the mechanics data in these six patients do not provide convincing evidence of a change in diaphragm activity, but do show the appearance of phasic inspiratory abdominal muscle activity and suggest an increase in intercostal and accessory muscle activity. In other circumstances where overall drive to breathe is increased, such as exercise and CO$_2$-stimulated breathing, abdominal muscles are recruited and intercostal and accessory muscles increase their activity.

Anesthesiology. V 82, No 6, Jun 1995
but this is normally associated with a considerable increase in activity of the diaphragm. Abdominal expiratory muscles do not show significant phasic expiratory activity with CO2-stimulated breathing until minute ventilation is about five times the resting level.24 By contrast, in our patients and others in whom abdominal muscle activity has been studied after surgery,16 the abdominal muscles have been found to be very active when the overall drive to breathe is only mildly increased. There is thus a shift in emphasis of respiratory neuromuscular output from the diaphragm to other major muscles of respiration.

Unusual Pattern of Breathing in Subjects 7 and 8

The postoperative pattern of breathing of the remaining two patients was dramatically different from the others. As shown in the Konno-Mead diagrams of figure 3, they shifted from a normal preoperative pattern to one in which abdominal motion was exaggerated and rib cage motion was greatly reduced. The pressure data, shown both as loops in figure 3 and pressure-time traces in figure 4, indicate that Pao2 actually decreased in inspiration. The changes in Pao2 increased slightly in magnitude and reversed in sign.

Because it is unreasonable to suppose that diaphragm muscle fibers are active through expiration and are deactivated in inspiration, the explanation of these data must be that there is a positive deflection in Pao2 at end-expiration as a result of passive stretching caused by contraction of abdominal muscles at a low lung volume. At the beginning of inspiration, abdominal muscles relax, releasing the passive stretch as the diaphragm descends and abdominal volume increases. In both subjects, Pao2 reaches its lowest value just after the end of inspiration. In subject 7, there is an increase in Pao2 at end-inspiration that must be attributed to active contraction of the diaphragm. Throughout inspiration, Pao2 in subject 7 thus has a decreasing positive component attributable to passive stretch, superimposed on a transient positive component attributable to active contraction. It is not possible to separate these components with precision but the amount of active Pao2 in this patient may be best estimated from the sudden drop in Pao2 when the diaphragm relaxes at a time when its length is changing little according to the rib cage abdomen plot. In subject 8, who showed no positive bump in Pao2 at end-inspiration, it is plausible to suppose that the diaphragm does not contract at all, being just passively stretched and released by abdominal muscles, but it is impossible to exclude some superimposed active component to Pao2 in inspiration. The pattern of breathing is similar to the one described for certain patients with bilateral diaphragm paralysis.25

Grunting Respiration

A curious pattern of pressure and motion during expiration was seen in the majority of patients on the 1st postoperative day. These patients apparently constricted their larynx or pharynx at end-inspiration to give themselves a partial upper airway obstruction and then relaxed their inspiratory muscles. They thus reverted to expiratory flow by adding an expiratory resistance instead of by prolonging inspiratory muscle activity into expiration. An example is shown in figure 5. After a normal inspiration, a plateau in volume begins at point A. During the plateau from A to B, pleural pressure shifts to a positive value and the rib cage volume increases while abdominal volume decreases. The shift in the rib cage–abdomen relation during the plateau probably signifies the return of the chest wall from a configuration distorted by inspiratory muscle tension to a relaxed configuration. There is a positive deflection in Pao2 at the same time as the one in pleural pressure but the amplitude of the gastric deflection was smaller because of rapid relaxation of the diaphragm occurring at the same time as the appearance of upper airway resistance.

This pattern of breathing corresponds to grunting respiration because when the airway is eventually opened, the positive end-expiratory pressure can cause a sudden, noisy expulsion of air. It is seen in newborn infants,26 in some patients with weak respiratory muscles,27 and in other patients with respiratory difficulties. It may help to keep mean lung volume increased in expiration at a comparatively low cost in terms of respiratory muscle work.

We found two types of breathing patterns among the eight patients observed after thoracic surgery. Six of the eight breathed in much the same way as patients described after abdominal surgery with a reduction in abdominal excursion relative to rib cage excursion and an increase in the ratio of changes in Pao2 to changes in Pao2. The detailed analysis of this pattern suggests it is caused by an increase in expiratory activity of abdominal or intercostal muscles and in inspiratory activity of intercostal muscles, with no increase in diaphragm activity. This pattern tends to reduce abdominal excursion and therefore probably reduce diaphragm excursion. The other two patients showed an increase in abdominal excursion relative to rib cage excursion, and appeared to be generating much of their tidal volume by contraction of abdominal muscles. In these cases contraction of the diaphragm was not enough to stabilize the flow, which continued to be driven by contraction of the abdominal muscles.

The latter type is sufficient to stabilize breathing in the supine patient, but any increase in it is simply another means by which patients can increase their minute ventilation and thus give rise to increased diaphragm tension. The diaphragm is simply another set of a pattern like this. It is an important mechanism in the respiratory system, and it may account for the variations in respiratory mechanics that we see in patients with respiratory difficulties.

The use of abdominal muscles in these patients is less than functional. In the second group with functional respiratory problems, a more extreme case may be where the abdominal muscles are so weak that they do not function at all. The grunting respiration may be an advantage in these patients, allowing them to maintain a better respiratory mechanics during expiration.

References


Anesthesiology. V 82, No 6, Jun 1995
by contraction and relaxation of abdominal muscles. In these cases diaphragm activity was probably reduced below the control level.

The latter two patients used intercostal muscles enough to stabilize the rib cage from collapse but not enough to expand it a great deal, and thus reduced motion of the rib cage. The other six patients breathed in such a way as to limit movement and pressure changes in the abdomen, and consequently to limit diaphragm motion.

Our observations do not give any information about the mechanism or consequences of the altered pattern of breathing in these patients. We speculate that the purpose of this is simply to immobilize the part of the chest wall that gives rise to the most activity from nociceptive receptors. The diaphragm may be the most sensitive region, accounting for the predominance after thoracic surgery of a pattern that like seen after abdominal surgery, but in some cases perhaps the rib cage compartment dominates in its requirement to be still. We could find no clinical corollaries for the different patterns.

The use of abdominal muscles in expiration in most of these patients implies that their end-expiratory volume is less than functional residual capacity, particularly in the second group. The magnitude of the decrease in functional residual capacity was not measured but in extreme cases may constitute most of the tidal volume. This change may have a detrimental effect on gas exchange. The grunting maneuver seen in several patients may be an advantage in this regard because it tends to prolong the time spent at end-inspiratory volume.

References